

[PubMed](#)[Nucleotide](#)[Protein](#)[Genome](#)[Structure](#)[PopSet](#)[Taxonomy](#)[OMIM](#)[Bc](#)Search 

for

[Limits](#)[Preview/Index](#)[History](#)[Clipboard](#)[Details](#)[About Entrez](#)[Text Version](#)[Entrez PubMed](#)[Overview](#)[Help | FAQ](#)[Tutorial](#)[New/Noteworthy](#)[PubMed Services](#)[Journal Browser](#)[MeSH Browser](#)[Single Citation Matcher](#)[Batch Citation Matcher](#)[Clinical Queries](#)[LinkOut](#)[Cubby](#)[Related Resources](#)[Order Documents](#)[NLM Gateway](#)[TOXNET](#)[Consumer Health](#)[Clinical Alerts](#)[ClinicalTrials.gov](#)[PubMed Central](#)[Privacy Policy](#)

☐ 1: Breast Cancer Res Treat 2000 Sep;63
(2):147-52

[Related Articles](#), [NEW Books](#),
[LinkOut](#)

Inhibitory effects of Indole-3-carbinol on invasion and migration in human breast cancer cells.

Meng Q, Goldberg ID, Rosen EM, Fan S.

Department of Radiation Oncology, Long Island Jewish Medical Center,
New Hyde Park, New York 11042, USA.

Indole-3-carbinol (I3C) is a promising phytochemical agent in chemoprevention of breast cancer. Our present study is the first description of I3C that significantly inhibits the cell adhesion, spreading and invasion associated with an up-regulation of PTEN (a tumor suppressor gene) and E-cadherin (a regulator of cell-cell adhesion) expression in T47-D human breast cancer cells. Therefore, I3C exhibits anti-cancer activities by suppressing breast tumor cell growth and metastatic spread. Metastatic breast cancer is a devastating problem, clinical application of I3C as a potent chemopreventive agent may be helpful in limiting breast cancer invasion and metastasis.

PMID: 11097090 [PubMed - indexed for MEDLINE]

[Write to the Help Desk](#)[NCBI | NLM | NIH](#)[Department of Health & Human Services](#)[Freedom of Information Act | Disclaimer](#)

sparc-sun-solaris2.8 Apr 15 2002 15:51:10



PubMed Nucleotide Protein Genome Structure PopSet Taxonomy OMIM Bc

Search PubMed



for

Go

Clear

☒ Limits

Preview/Index

History

Clipboard

Details

About Entrez

Display

Abstract

Sort

Save

Text

Clip Add

Order

Text Version

Entrez PubMed

Overview

Help | FAQ

Tutorial

New/Noteworthy

PubMed Services

Journal Browser

MeSH Browser

Single Citation Matcher

Batch Citation Matcher

Clinical Queries

LinkOut

Cubby

Related Resources

Order Documents

NLM Gateway

TOXNET

Consumer Health

Clinical Alerts

ClinicalTrials.gov

PubMed Central

Privacy Policy

☐ 1: Biochim Biophys Acta 2000 Dec 20;1498(2-3):99-111Related Articles, ^{NEW} Books, LinkOut**ELSEVIER SCIENCE
FULL-TEXT ARTICLE**

The biology of the receptor for advanced glycation end products and its ligands.

Schmidt AM, Yan SD, Yan SF, Stern DM.

Department of Surgery, College of Physicians and Surgeons of Columbia University, New York, NY 10032, USA.

Receptor for advanced glycation end products (RAGE) is a multiligand member of the immunoglobulin superfamily of cell surface molecules whose repertoire of ligands includes advanced glycation end products (AGEs), amyloid fibrils, amphoterins and S100/calgranulins. The overlapping distribution of these ligands and cells overexpressing RAGE results in sustained receptor expression which is magnified via the apparent capacity of ligands to upregulate the receptor. We hypothesize that RAGE-ligand interaction is a propagation factor in a range of chronic disorders, based on the enhanced accumulation of the ligands in diseased tissues. For example, increased levels of AGEs in diabetes and renal insufficiency, amyloid fibrils in Alzheimer's disease brain, amphoterin in tumors and S100/calgranulins at sites of inflammation have been identified. The engagement of RAGE by its ligands can be considered the 'first hit' in a two-stage model, in which the second phase of cellular perturbation is mediated by superimposed accumulation of modified lipoproteins (in atherosclerosis), invading bacterial pathogens, ischemic stress and other factors. Taken together, these 'two hits' eventuate in a cellular response with a propensity towards tissue destruction rather than resolution of the offending pathogenic stimulus. Experimental data are cited regarding this hypothesis, though further studies will be required, especially with selective low molecular weight inhibitors of RAGE and RAGE knockout mice, to obtain additional proof in support of our concept.

Publication Types:

- Review
- Review, Tutorial

PMID: 11108954 [PubMed - indexed for MEDLINE]